

Too Dangerous to Dip? Marine Pollution Makes Swimmers Sick

Reports of illness after exposure to marine water appear to be increasing, and there is evidence that the rate of infection is proportional to both the amount of time swimmers are exposed and the levels of pollution in the waters where they swim. In this issue, researchers from Harvard University led by Sarah E. Henrickson conclude that human activities are contributing to illnesses associated with marine recreation [*EHP* 109:645–650]. The team also suggests ways to reduce the incidence of illness by both monitoring pathogens to prevent human exposure and correcting environmental conditions that boost pathogen concentrations in marine waters.

Marine pollution is increasing: up to 90% of untreated sewage from urban areas in developing nations is dumped into oceans and streams, and high nitrogen loads from agricultural runoff and other types of pollution may upset the ecological balances that normally keep pathogens in check. Warming oceans and extreme weather events also contribute to the growth of algal blooms that can harbor pathogens.

Research reviewed by the Harvard team suggests that bacterial pathogens can survive in salt water, especially in waters with low salt concentrations (such as those found in estuaries) or high nitrogen loads. For example, marine species of *Vibrio* bacteria have been linked to disease in swimmers, including cases of gangrene and endometritis. Outbreaks of noninfectious disease are also of concern. These include various types of “swimmer’s itch” caused by marine larvae, and neurologic symptoms from exposure to algal toxins. Swimmers can be exposed to pathogens through broken and unbroken skin. Exposure to algal toxins can also occur through the lungs when the toxins are aerosolized by heavy surf.

Although public health authorities most often test for bacterial contamination, the authors’ review of 131 articles suggests that the primary health hazard in coastal waters may not, in fact, be bacterial exposure, but viral instead. The authors examine, for example, one randomized controlled trial of swimming exposure conducted in the United Kingdom in which swimmers became ill even on days when concentrations of fecal coliform bacteria were at acceptable levels.

Viruses are extremely abundant in marine waters, where they often survive longer than bacteria. They are also more resistant to sewage treatment. The authors speculate that viruses may be a chief cause of swimming-associated gastrointestinal infections.

The Harvard team concludes that bacterial indicators are inadequate surrogates for overall pathogen levels and that surveillance methods that focus only on these indicators are no longer adequate to protect human health. They recommend the development of surveillance methods using polymerase chain reaction, fluorescent antibody, or monoclonal antibody techniques. They also recommend monitoring algal populations. To reduce the number of pathogens in marine waters, they recommend reducing nutrient loads through maintenance of wetlands that filter runoff, an increase in the efficiency of sewage treatment and other urban waste disposal systems, and reductions in the use of fertilizers. They cite the example of Sydney, Australia, which greatly reduced health hazards at its beaches by extending sewage outfalls to the edge of the continental shelf. The authors also state that reducing emissions from fossil fuel combustion would decrease the amount of nitrogen deposited in marine ecosystems and help prevent long-term warming of the world’s oceans.
–Kris Freeman

Funeral Wreaths Brazilian Deaths Traced to Common Marine Toxins

Beginning in February 1996, 131 patients who had been treated that month at a dialysis center in the Brazilian city of Caruaru developed symptoms including headache, nausea, vomiting, and blurred vision. One hundred subsequently suffered acute liver failure, and 76 died. Of these 76 victims, 52 deaths are attributed to the cyanotoxin-related disease now called Caruaru syndrome. The waterborne cyanobacterial toxins that killed those 52 patients are likely to claim more lives worldwide as reservoirs become increasingly polluted, warns a team of researchers studying the Brazilian outbreak, the first documented human cases of Caruaru syndrome [*EHP* 109:663–668]. “Since many of the world’s reservoir- and lake-based water supplies are subject to increasing levels of nutrients, it is highly probable that repeat episodes of cyanotoxin poisoning will occur unless measures are taken to better understand [cyanobacteria’s] role in water-based disease,” the team writes.

Although they are technically bacteria, cyanobacteria behave like waterborne algae. The toxic cyanobacteria linked to Caruaru syndrome originated in an algal bloom on the Tabocas Reservoir, which supplies Caruaru’s water. According to study leader Wayne Carmichael of Wright State University in Dayton, Ohio, toxic blooms are found on many of the world’s surface waters. The 40 species of known or suspected toxin-producing cyanobacteria produce six chemical groups of toxins.

Water treatment normally removes toxic cells or deactivates the toxins, but the water used at the dialysis center had not been fully treated. To avoid chlorine residue in the water used for dialysis, the center had trucked water in from the municipal plant after it was treated only with alum flocculation. The dialysis center then further treated the water at its own in-house plant, but the poorly maintained plant failed to remove the cyanotoxins.

Brazilian authorities initially attributed the outbreak to chemical contamination of the water used in dialysis treatment. However, Sandra Azevedo of the University of Brazil in Rio de Janeiro suspected cyanobacterial toxins were the real culprit when the patients died quickly without developing any of the neurologic symptoms associated



Swim for shore! Various contaminants in polluted swimming areas can cause bacterial and viral infections, skin problems, or neurologic symptoms.

with chemical poisoning. She, Carmichael, and an international team of researchers traced the syndrome to intravenous exposure to cyanobacterial toxins.

By examining water samples and patients' serum and liver tissue, the researchers identified the major toxin group responsible as hepatotoxic microcystins. They estimated that microcystin concentrations in the water used for dialysis were 19.5 micrograms per liter, nearly 20 times the limit proposed by the World Health Organization as safe for drinking water. The researchers first reported those findings in the 26 March 1998 issue of the *New England Journal of Medicine*.

Since then, they discovered that the water used in dialysis treatment also was contaminated with the cyanotoxin cylindrospermopsin, which has been linked to an outbreak of liver and kidney disease among 148 people in Australia. Carmichael and his colleagues had suspected that cylindrospermopsin was in the Caruaru water because the organism that produces this cyanotoxin was identified in water samples from the Tabocas Reservoir. To date, however, the researchers have not identified cylindrospermopsin in the Caruaru liver specimens. But that's probably because the specimens were prepared specifically for microcystin analyses, the researchers admit.

Carmichael says he and his colleagues recently developed a new method of measuring cylindrospermopsin in liver samples and hope to test it soon. "We want to be able to predict as closely as possible what levels of cylindrospermopsin and microcystins it took to kill these dialysis patients," he says. The researchers call for public officials to help prevent more outbreaks of Caruaru syndrome by protecting surface water supplies. —Cynthia Washam

Frequent Fumers

Airport Study Shows Smoke Gets to Your Heart

If you want to give yourself a real heartache, spend about two hours in one of those glass-enclosed smoking areas at some of the nation's airports. Healthy nonsmoking volunteers working with researchers from Brigham Young University, Harvard University, and the University of Utah spent equal time in the smoky cubicle at Salt Lake International Airport and a smoke-free section of the airport [*EHP* 109:711–716]. The researchers measured the volunteers' heart rate variability—a marker of the health of the autonomic function of the heart—and found that sitting in the smoking chamber adversely affected this function of the heart.

"The most significant finding of our research is that we have observed that a relatively low concentration of tobacco smoke particles is associated with declines in heart function that may lead to adverse cardiac events," says lead researcher C. Arden Pope, III, a Brigham Young professor of economics who studies the impact of the environment on economic issues. Pope says the work may lead to the identification of the mechanistic pathways that allow particles from combustion to manifest as increased risk to the heart.

"To my knowledge," Pope says, "this is the first time we have been able to show that environmental tobacco smoke—[ETS, or] passive smoke—creates changes in heart rate variability." Pope says heart rate variability, the physiologic response of the heart to different activity levels, is well-described in the medical literature as a cardiac risk factor. Some researchers have suggested that failure of the heart to respond rapidly to changing circumstances bodes ill for long-term health, so reductions

in heart rate variability caused by ETS exposure could prove detrimental (although this area is still controversial).

For many years scientists have seen an association between ETS and heart problems, but Pope and his colleagues saw that the physiologic pathways that turn ETS exposure into heart trouble had been left largely uncharted. In 1999 they set out to address this information gap. The researchers enrolled 16 adults aged 21–75. Nine men and seven women were divided into two eight-person panels. The subjects were hooked up to ambulatory heart monitors and sent into either the glass-enclosed smoking lounge at the airport or a nonsmoking area with a similar setting. Each panel completed two two-hour stints in both the smoking and nonsmoking areas. The participants would have been allowed to leave the test if they became ill or uncomfortable; however, all subjects completed the eight-hour length of the study.

The researchers monitored the blood oxygen concentrations of the subjects at regular intervals throughout the study. They also charted heart rate variability associated with standing, sitting, eating, and use of the bathroom. The subjects kept detailed activity diaries, which were used to rule out other possible activities involved in changes in heart rate.

The team found that during the smoking-lounge periods subjects experienced an average of a 12% decrement in the standard deviation of all normal-to-normal heartbeat intervals, which measures the time between specific points in a heartbeat and provides an estimate of overall heart rate variability. In other words, the subjects' heart rate variability diminished when they were being exposed to ETS. "This indicates a pathophysiological link between chronic environmental tobacco smoke exposure and cardiovascular health through the autonomic workings of the heart," Pope says.

Pope says this study correlates well with other studies showing that outside air pollutants have effects on the autonomic system. He also says that exactly what particles in smoke are responsible for the effect on the autonomic system requires further study. —Ed Susman



Breathing room needed. Heart rate variability among subjects stationed in an airport smoking lounge shows that a relatively low concentration of tobacco smoke particles is associated with declines in heart function.